Extensive Tissue Necrosis Following High-Concentration Sclerotherapy for Varicose Veins

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BACKGROUND. Tissue necrosis after sclerotherapy has been observed, but is unexplained.

OBJECTIVE. To present the complication of extensive tissue necrosis following high-concentration sclerotherapy for varicose veins.

METHODS. Cases coming to the attention of the authors are presented briefly with commentary and discussion to explain the mechanisms of tissue destruction.

RESULTS. Although the complication of extensive tissue necrosis has been ascribed to intra-arterial injection, in fact, careful study of the cases described here shows that intravenous injection was present in each case. A theory of distribution of the sclerosant into the arterial arborization is proposed. This theory would explain the distribution of sclerosant into the arterial tree and would also explain the causation of extensive tissue necrosis. Mention is made of experimental work in which intra-arterial injection was not the mechanism of causation of tissue necrosis.

CONCLUSION. Extensive tissue necrosis following high-concentration sclerotherapy may be rare, but its occurrence is serious and its treatment may be incomplete.

A RESURGENCE of interest in treating venous varicosities has resulted in increased utilization of sclerotherapy. In English-language countries, the techniques developed by Fegan at the St. James' Hospital in London have been dominant.[1] On the continent, different techniques were developed by Tournay[2] and Sigg.[3] Some doubt as to the efficacy of the Fegan sclerotherapy was published by Hobbs,[4] but it has been duplex ultrasound which has allowed evaluation of sclerotherapy in total treatment of truncal varicose veins.[5] Bishop et al.,[5] in studying patients treated by Fronek without ultrasound guidance, concluded that the presence of hemodynamically significant reflux originating at the level of the saphenofemoral junction caused failure of sclerotherapy treatment of the greater saphenous vein. Other reports using duplex monitoring of attempted sclerosis of the greater saphenous vein have confirmed those findings.[6]

The technique of sclerotherapy of the greater saphenous vein demands high-concentration sclerosants. Ultrasound-guided sclerotherapy attempts to increase the safety of this procedure. A particularly well-monitored study from Berlin confirmed that sclerosis of the greater saphenous vein without ultrasound guidance using concentrated solutions was effective in 52% of limbs at 3 years.[7] Greater saphenous reflux recurred in 75% of limbs with deep venous reflux and in 33% of limbs without deep venous reflux. If greater saphenous veins had no reflux at 3 years, there was no reflux for at least 5 years of observation.

A study was undertaken to compare two methods of sclerosing the saphenofemoral junction (SFJ) without duplex ultrasound.[8] The study involved a sample of 2186 patients. In the first method, with the patient in the sitting position, injections were administered below the SFJ with digital compression applied directly to the SFJ. In the second method, with the patient in the standing position, injections were isolated to below the junction, with digital compression applied directly to the junction as well as below the injection site. Results showed that this method of sequestration of the proximal saphenous segment with the patient in a standing position produces a valid technique for the treatment by sclerosing injections of incompetence of the long saphenous vein.[9]

The possibilities of ultrasound-guided sclerotherapy using increasingly concentrated solutions have been further explored and are reported to have few side effects. Most recent studies include those of Kanter and Thibault,[10] who reported their experience with saphenofemoral incompetence treated by ultrasound-guided sclerotherapy. They reported 202 limbs treated with 3% sodium tetradecyl sulfate. Forty-eight limbs (23.8%)

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were found by duplex ultrasound to have recanalized at 1 year. However, 0 of 28 (0%) at 2 years were recanalized (cumulative rate 48/202; 23.8%). More importantly, they reported no extensive tissue necrosis or other complications. Similarly, Thibault and Lewis[11] reported the safety of treating incompetent perforating veins. In France, Miserey et al.[12] reported that more than 2500 injections of the saphenofemoral and saphenopopliteal junction were performed by them under ultrasound guidance without complications.

However, Biegeleisen et al.[13] reported on ultrasound-guided injections which led to tissue necrosis and ascribed this to inadvertent arterial injection. They concluded that the medical literature does not contain enough data to enable one to formulate a definitive strategy either for prevention or for treatment of this complication.

A number of cases of extensive soft tissue necrosis have come to our attention over the last few years. We believe that this major complication in use of concentrated sclerosing agents with or without ultrasound guidance should receive attention. The following case reports are presented to illustrate the unpredictable nature of this occurrence even when done by physicians with considerable experience.

Case Reports

Patient 1

A 43-year-old man had removal of symptomatic varicose veins by ankle-to-groin surgical stripping. At 6 weeks postoperatively, a medial ankle varix was injected with 3% sodium tetradecyl sulfate by an experienced vascular surgeon. Immediate pain, foot blanching, and pronounced dysesthesias led to arteriography. Marked spasm without occlusion was present in the posterior tibial artery distribution. Intra-arterial nitroglycerin, papaverine, and bolus unfractionated heparin was administered. Extensive tissue necrosis corresponding to the distribution of the lateral plantar artery developed (Figure 1). An above-knee amputation was done.

This case is similar to others reported in the pre-duplex ultrasound era. The operator, usually experienced, attempts varix sclerosis using a concentrated solution. Immediate pain, pallor, paresthesias, and subsequent paralysis precede soft tissue necrosis. As the clinical picture is that of an acute arterial occlusion, this is given as the most likely cause of the unfortunate event.

Patient 2

A 36-year-old man had a family history of varicose veins. His brother had bilateral vein surgery at age 19. Physical examination was unremarkable except for numerous tortuous varicosities in tributaries to the greater saphenous system bilaterally. There was no stasis dermatitis or ulceration. In initial treatment, the patient had multiple injections of varicosities using 3% sodium tetradecyl. There was immediate posterior tibial neuralgia that responded to local injections of 1% xylocaine. The relief was temporary, and very severe pain in the foot developed as the local anesthetic effect wore off. There was some cyanosis over the medial aspect of the foot, although capillary refill was seen. There was no motor nerve dysfunction, but there was hypersensitivity over the medial aspect of the plantar surface. Local infiltration of 1% xylocaine

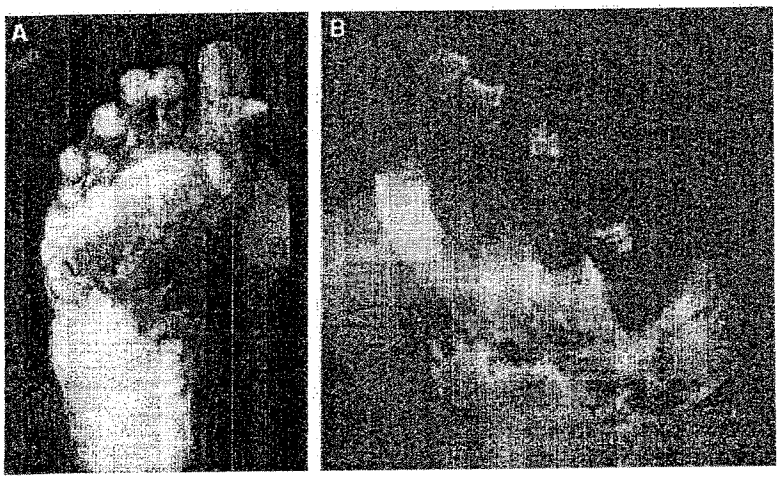

Figure 1. A) Plantar view of the area of tissue necrosis in patient 1 outlines the posterior tibial artery distribution which led to the diagnosis of inadvertent intra-arterial injection. B) Necrosis extending to the dorsal as well as the plantar aspect of the distal forefoot.

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was again instilled and the patient was admitted to the hospital.

The posterior tibial artery was cannulated and heparinized saline infused. A hemostatic tourniquet was inflated as 250,000 U of urokinase were infused in the foot. The infusion was repeated three times and between each of the infusions, papaverine was infused using a total of 60 mg of papaverine in divided doses. Immediate hyperemia occurred with violaceous, erythematous changes in the skin of the foot and base of the toes. A few small mottled areas remained on the medial aspect of the foot and these eventuated into patchy necrosis.

Six months later, because of contracture of the flexor hallucis brevis muscle and plantar fascia, surgical exploration was undertaken. This revealed extensive scarring around the posterior tibial nerve, especially in the tarsal tunnel at the ankle. The medial and lateral plantar nerves were identified and as they were traced distally, they were found to be quite compressed by the scarred body of the flexor hallucis brevis muscle. Marked scarring of the flexor hallucis brevis muscle was seen. The medial aspect of the plantar fascia was found to be very tight.

Intra-arterial injection is highly unlikely in this situation in which isolated flexor hallucis muscle necrosis occurred. A pure intra-arterial injection would have to have been directly into the medial branch of the posterior tibial artery. This would be impossible with a simple subcutaneous injection.

It is apparent that intra-arterial flushing with heparinized saline, administration of papaverine as a vasodilator, and infusion of urokinase as a thrombolytic agent was effective in salvaging most of the soft tissue of the foot and preventing extensive soft tissue necrosis. Flexor hallucis brevis necrosis and contracture produced considerable disability.

Patient 3

A 65-year-old woman complaining of calf pain was found to have venous insufficiency of the lesser saphenous and gastrocnemius veins. For symptomatic relief, 0.5 cc of 3% sodium tetradecyl sulfate was injected into the lesser saphenous vein approximately 5 cm below the saphenopopliteal junction under duplex guidance. Slight pain was experienced immediately following the injection. This soon subsided. No blanching was observed. No sharp excruciating pain or paresthesia was felt. Five hours later the patient complained of a footdrop of new onset. Duplex examination revealed no abnormalities. Over several days, a violaceous discoloration developed in the popliteal fossa and upper calf. Breakdown of skin commenced 10 days after the initial injection in the lesser saphenous vein location. A prolonged course of tissue necrosis and debridements required months to heal. Peroneal and posterior tibial nerve injury persisted. With skin grafting and physical therapy, a near complete recovery was obtained over 18 months.

Injections into the area of the saphenopopliteal junction are particularly hazardous. This case shows that the hazard is diminished but not eliminated by an experienced sclerotherapist working with an excellent duplex technologist. As the injection in this case was clearly intravenous at all times, a theory of origin of tissue necrosis apart from inadvertent arterial injection must be constructed.

Patient 4

A 38-year-old woman had undergone numerous sclerotherapy treatments in the past for cosmetically unacceptable varicose veins. Because of the development of new symptoms and clinically apparent varicose veins, ambulatory phlebectomy was also performed. Additional sclerotherapy treatments were then performed for cosmetic concerns both with and without ultrasound guidance. The last sclerotherapy session with 2% sodium tetradecyl sulfate produced a porcelain-white blanching of the skin immediately after injection. Nitroglycerin paste was rubbed into the affected area in an attempt to relieve vasospasm. A class II graduated compression stocking (25.1–32.1 mmHg pressure) was applied and the patient was sent home.

In this case, the injection was most likely into a tributary of the lesser saphenous vein. The duplex ultrasound findings were clear and the dose of concentrated sclerosant was small. However, the wide distribution of tissue damage suggests an arterial distribution of the sclerosant.

One week later when the patient removed the stocking, she noticed a large area of blue discoloration on the medial calf at the point of injection. Physical examination showed a 7 cm × 5 cm, dusky blue patch on the medial calf with two areas, 5 mm in diameter, of superficial necrotic skin (Figure 2). The patient was given pentoxifylline 400 mg three times a day. When seen 1 week later, the large area of dusky blue skin was normalizing but the superficial necrotic skin persisted.

In this instance, the injection was again clearly intravenous even though the effects were those of arterial ischemia. The constant wearing of the compression stocking precluded detection and treatment of the color changes.

Patient 5

In this case a duplex-guided injection of 1 cc of 3% sodium tetradecyl sulfate was done into an incompetent gastrocnemius vein. The patient's problem had been posterior calf telangiectasias without symptoms,

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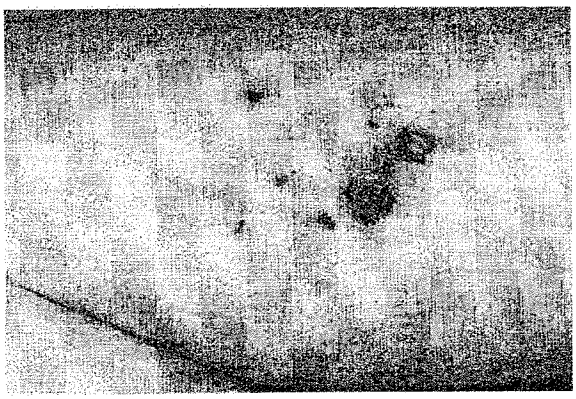

Figure 2. The resolution of the area of compromise following successful treatment. The large area of bluish discoloration resolved from 7 cm × 5 cm in size to two areas 5 mm and 7 mm in diameter as shown here.

and no associated varicosities were noted. Immediate pain required treatment with intramuscular meperidine. Two days later the entire posterior calf assumed a mottled blue discoloration. Three weeks later a well-demarcated necrotic area was apparent that was subsequently debrided down to muscle fascia (Figure 3). A full-thickness skin graft was applied with poor cosmetic results. A persistent neurologic deficit resulted in a dysfunctional gait.

Sclerotherapy of the lesser saphenous territory using high-concentration solutions can be dangerous, but another lesson to be learned from this case is to treat the appropriate lesion. At times the duplex may demonstrate a deep vein that is seen to be incompetent. However, in the absence of demonstrated significant reflux into the surface blemish, it is most likely that sclerotherapy of the gastrocnemius veins was unnecessary. Treatment of the telangiectasias was all that was required. When using concentrated sclerosants, one must listen to the patient's complaint of pain. In surgery, one does not treat appendicitis with opiates. In sclerotherapy, one should not treat the symptoms of pain, but instead should determine the reason for the pain. Early intervention on the effects of sclerotherapy may minimize damage and possibly prevent tragedy.

Discussion

These cases demonstrate the potential hazard of high-concentration sclerosant injection when used in the treatment of distal varicosities and major veins. Four of the injections described were directed into large veins and one into a very small varix. These cases occurred at the hands of experienced physicians who followed correct procedures.

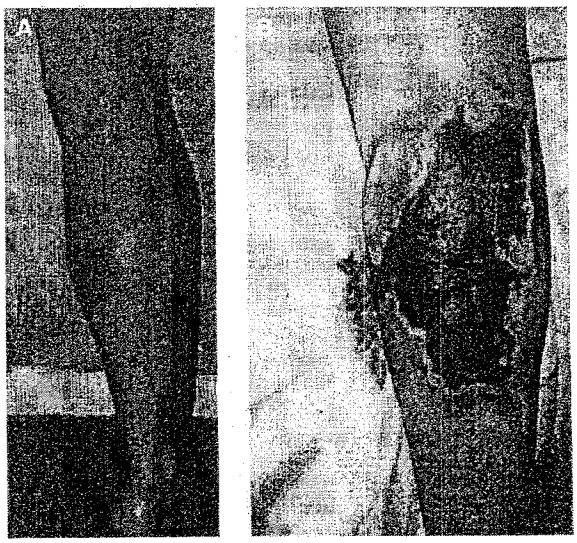

Figure 3. A) Mottled skin 48 hours after injection of 1% sodium tetradecyl sulfate into the gastrocnemius vein. B) The well-circumscribed necrotic area is apparent 3 weeks after initiation of the tissue necrosis by sclerotherapy. (From Figure 8.24 in Sclerotherapy, 2nd ed. Goldman MP, ed. St. Louis C.V. Mosby, 1998:327; with permission).

Prior to 1986, several reports pointed out the dangers of major vein sclerosis using high-concentration sclerosants. Cockett[14] reported 18 such cases with two major amputations, 12 transmetatarsal amputations, and 14 other patients seriously invalided. Natali,[15] reporting from Paris, reported eight accidents after injection of sclerosants into the greater saphenous vein during a 3-year period ending in 1985. In the next 3 years, seven other cases came to Natali's attention and two major amputations were required. Eight other patients experienced extensive muscle necrosis and others had variable amounts of cutaneous scarring with or without attendant muscle damage.

More recent reports[16–18] have focused on long-term results in correction of reflux in the greater saphenous vein and have not reported serious complications such as those detailed above. In contrast, Grondin et al.[18] called attention to serious complications of high-concentration sclerotherapy at the hands of the developer of the method, R. M. Knight, after he presented his technique at the annual meeting of the Phlebology Society of America in 1991.[19]

Natali called attention to the medicolegal implications of major complications of sclerosis, pointing out that serious accidents occur more readily after attempted sclerosis of the lesser saphenous vein at its termination. He also called attention to necrosis in the inguinal area after sclerosis of the greater saphenous vein near the saphenofemoral junction.

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Saliou et al.[20] reported thrombosis of the peroneal artery demonstrated by arteriogram after injection of polidocanol 0.5% into the popliteal fossa in attempted sclerosis of the small saphenous vein. Immediately after the injection, violent pain occurred in the calf and the foot became pale and numb. Intravascular vasodilators were ineffective, and cutaneous necrosis became apparent 24 hours later. A three-compartment fasciotomy was performed. Multiple debridements were required before reconstructive surgery was performed 3 months later.

MacGowan et al.[21] investigated intra-arterial injection of 3% sodium tetradecyl in experimental animals. Their extensive studies involved greyhound dogs, a Perspex ear chamber in rabbits, and further experiments with New Zealand white rabbits. The results of those experiments indicated that sodium tetradecyl, when injected intra-arterially, had little effect on the major vessels, produced no spasm, but was washed into smaller arteries and arterioles where blood was converted into a thick lysate or sludge. The sludge acted as an embolus to obstruct the microcirculation, causing proximal stagnation, secondary thrombosis, and necrosis of tissue. In those experiments there was no evidence that intimal irritation or damage caused the changes, but instead that mechanical blockage took place.

In many clinical cases it is clear that arteriospasm plays a major role in contributing to soft tissue ischemia. This is manifest as visible pallor and, in the few cases in which arteriography has been performed, the spasm is evident on the arteriogram as in patient 1 above.

Reports cited in the discussion above assume direct arterial injection as the cause of extensive tissue necrosis. However, another explanation may be valid. de Takats,[22] emphasizing the studies of Burch, describes and illustrates preferential arterial and venous connections in the precapillary circulation (Figure 4) stating that, "These shunts permit more direct transmission of pressure from the arterial to the venous system. They offer less resistance to flow than the capillaries and help maintain venous pressure and flow." In the situation of prolonged chronic venous hypertension which has caused elongation, dilation, and valve incompetence in lower extremity veins, free flow of blood from the venous system to the arterial system is easy to contemplate.

Goldman[23] has called attention to the fact that "even when sclerotherapy is performed with expert technique, using the safest sclerosing solutions and concentrations, cutaneous ulceration may occur. Therefore, it appears that extravasation of caustic sclerosing solutions alone is not totally responsible for this complication." Goldman further points out that histologic

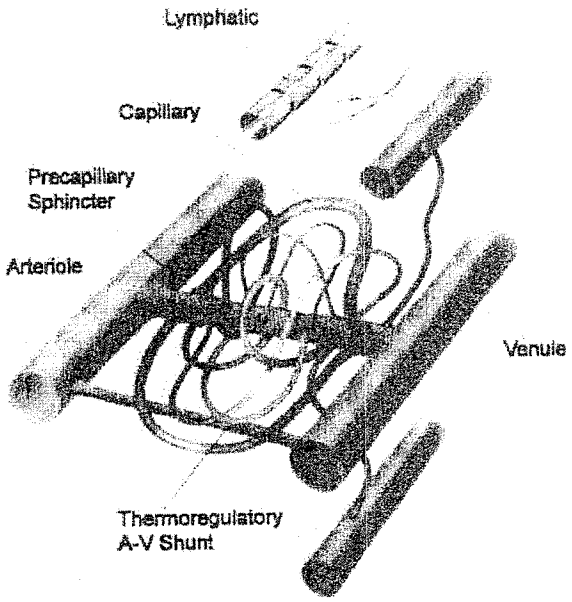

Figure 4. The intimate relationship of arterioles, venules, and thermoregulatory shunts is shown in this drawing. It is easy to understand that chronic venous hypertension can produce failure of precapillary sphincters, thus allowing arterial blood to flow into the venous system. Likewise, injections into venules stretched by chronic venous hypertension may allow passage of the sclerosant into the arterial system for distribution along the pathway of a major artery. (From Phlebolymphology, Servier et Cie; with permission).

examination of excised cutaneous ulcerations reveals occlusion of the feeding dermal arteriole in each case. Occlusion of the arteriole produces wedge-shaped tissue necrosis, a classical infarct. These observations suggest that, as seen in Figure 4, chronic venous hypertension may open arteriovenous thermoregulatory shunts, allowing sclerosant to pass directly into the arterial side of the circulation despite meticulous intravenous injection of the concentrated agent.

Goldman[24] has described an immediate postinjection, porcelain-white appearance of the skin during intravenous injection, suggesting profound spasm of the arterial arborization. Such an observation has been ascribed to intra-arterial injection. However, proof of direct intra-arterial injection of sclerosant is lacking. In fact, directly confirmed intravenous injection by duplex ultrasound guidance of the sclerosant is the rule rather than the exception. Observation of the shape and distribution of the area of necrosis produced by the injection is circumstantial evidence of that suggests intra-arterial injection.

The most common location for so-called arterial injection is in the region of the Cockett perforating veins

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in the retromalleolar ankle area, and attention has been called to the fact that this is particularly close to the posterior tibial arterial circulation. Pain is usually noted immediately and this pain propagates in the distribution of the lateral plantar artery into the foot and fourth and fifth toes. This is similar to patient 2 above. Pedal pulses may remain present, but within 10–12 hours, severe blanching of the fourth and fifth toes is seen, and the sole of the foot becomes painful. Such progression is in the distribution of the plantar arteries and suggests that the injection is intra-arterial. Nevertheless, physicians, often experienced sclerotherapists, have documented to their satisfaction the injection was intravenous despite the arterial distribution of the complication.

Biegeleisen et al.[13] reported that as little as 1 ml of 3% sodium tetradecyl sulfate caused necrosis. Four of their cases involved necrosis in the distribution of the medial superficial sural artery. They point out that in at least one case, the injection was under ultrasound guidance and the needle "appeared to be unequivocally intraluminal and intravenous." The color photograph accompanying the presentation shows posterior calf skin necrosis which resulted in the loss of 3–5 cm^2 of skin. In commenting on prevention of necrosis following high-concentration sclerotherapy, they said, "Because all our cases resulted from injections into the proximal greater or lesser saphenous veins, eliminating these injections would certainly reduce the number of accidents."

We have found that virtually any injected area can be hazardous, as in patient 4. Ulceration in an arterial distribution has been seen to occur in the posterior thigh, the midthigh, and the midcalf, in addition to the acknowledged areas at the medial malleolus and inner thigh. In the cases described above, most tissue damage appears to occur with injection of the saphenopopliteal junction.

Biegeleisen et al.[13] calls attention to the fact that there are pitfalls in ultrasound image interpretation. Important structures such as the femoral artery may look to be farther away than they actually are. The problem occurs with imaging a three-dimensional structure in two dimensions. A needle that appears in the plane of a targeted vein may be anterior or posterior to it. We suggest that prior to commencing injection, a small bolus of air may be injected ahead of the sclerosant. Alternatively, a few drops of solution may be injected and the patient interrogated regarding pain. Some sclerotherapists advocate using an open-needle technique to ensure that an arterial puncture has not occurred. However, dangers of viral contamination preclude general use of this method. Also, careful observation for distention around the injection site should be done. Even with these safeguards, the purely intravenous distribution of the sclerosant cannot be assured.

Therapeutic efforts to treat apparent arterial injection of sclerosant are usually unsatisfactory but should be attempted. Arterial injection should be suspected if severe pain accompanies cutaneous pallor. Unfortunately, some patients will have no complaint of immediate pain and will only demonstrate a mild, sharply demarcated erythema which becomes dusky and cyanotic after a few hours to days.

Possible treatments include infiltration of the affected area with procaine, which forms a complex with sodium tetradecyl sulfate and renders it inactive.[25,26] The area should be cooled with ice packs to minimize tissue anoxia by decreasing tissue metabolism. However, this may cause local vasoconstriction. Immediate heparinization with fractionated heparin should be given and continued for 6 days. Some consideration should be given to an immediate bolus of unfractionated heparin intravenously. Administration of low molecular weight dextran at a rate of 10 ml/kg for 3 days may be attempted. In severe cases, arteriography and installation of vasodilators may be indicated. Use of a thrombolytic agent may also be considered. Finally, use of oral prazosin, hydralazine, or nifedipine for 30 days may also be considered.

In one clinical report, use of intravenous heparin followed by subcutaneous heparin was found to be effective in averting skin necrosis.[13] It has been observed that warfarin is not as effective as subcutaneous heparin in patients with tissue necrosis after sclerotherapy.

As treatment of this complication is unlikely to yield very satisfactory results, we recommend that sclerotherapy for varicosities and duplex ultrasound-guided sclerotherapy be performed only by experienced physicians. Areas which have led to the most tissue damage, such as the saphenopopliteal junction, should be approached with the utmost caution. No technique guarantees complete safety. Unexpected and unpredictable venous-to-arterial connections in the precapillary circulation may allow intra-arterial passage of sclerosant from its initial venous entry.

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Commentary

This succinct and scholarly monograph authored by internationally recognized phlebologists may well represent the most comprehensive and eminently practical analysis of this enigmatic complication to date. The importance of such an article is derived as much from the fundamental questions it raises as the information (historical, statistical, clinical, phlebological and anatomical) it presents. Here we have a procedure whose success requires the use of tissue toxic concentrations of sclerosants which despite sophisticated technology (duplex guidance) and flawless technique can result in completely unpredictable and horrific complications for which no good treatment exists.

Several questions come immediately to mind:

1. Does the rarity of severe complications justify the use of high concentrations of sclerosants? The notion that "experts" can prevent these complications seems negated by the clinical data.
2. Will radio-frequency ablation or refinements in surgical technique provide safer alternatives? In the case of junctional incompetence, I am more comfortable with the direct visualization afforded by surgical approaches; although in the case of saphenopopliteal incompetence, anatomical complexities make surgery extremely difficult.
3. Could a careful analysis of the circumstances associated with these serious complications be of value in establishing specific treatment guidelines, ie, should this procedure be carried out in certain anatomical areas or in the presence of chronic venous hypertension?
4. Can tissue necrosis occur without arteriolar sludging? We have observed what appears to be direct cytotoxic effects following injection of 1% Sotradecol, hypertonic saline and sodium morrhuate intradermally on the bicep of a volunteer. As an aside, we have demonstrated increased vascular fragility in patients over 60 years of age resulting in ulceration using low concentrations of sclerocants.[1] Finally, I don't believe for one minute that .5% polidocanol (the equivalent of .2% Sotradecol) can produce massive tissue necrosis. I suspect that 5%, not .5% polidocanol, was employed.

In conclusion, this is a superb and provocative article which clearly describes the details, scope and complexity of the issues surrounding the use of high concentration sclerosants.

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Commentary

Complications that I have seen in practice when high-concentration sclerosants have been used include severe pain, inflammation, and hyperpigmentation. I certainly agree that this is not the way to treat venous insufficiency. A case in point illustrates this quite nicely and supplements the information in the present manuscript.

A 25-year-old woman with one child and with a strong family history of varicose veins had been troubled by large veins in the right lower extremity for seven years. Saphenofemoral incompetence was present with reflux to calf level. No diameter of the greater saphenous vein was noted (Fig. 5A). Ultrasound-guided sclerotherapy was decided upon and after a test dose of 1.5 ml of 3% sodium tetradecyl sulfate was given, a definitive

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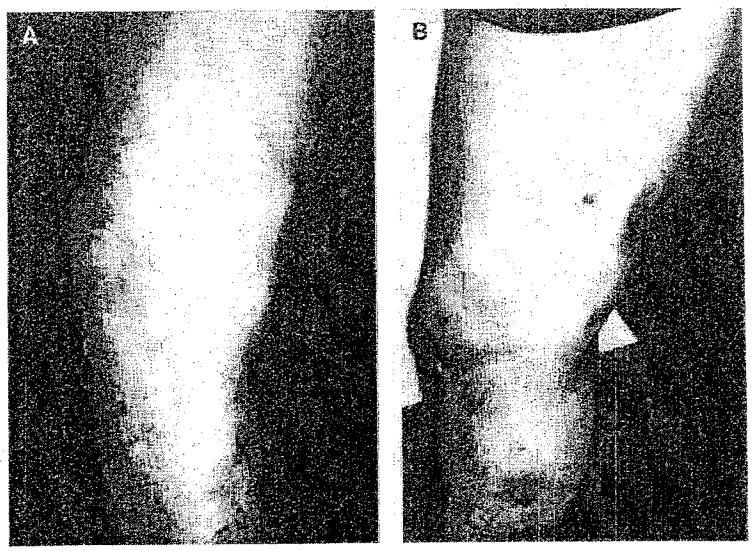

Figure 5. A) The dilated and tortuous saphenous vein as well as the dilated and tortuous anterior tributary veins. B) After extensive skin breakdown, abscess formation, and healing by secondary intention, the end result was the unsatisfactory scars shown here.

ultrasound-guided sclerotherapy session was scheduled for two weeks later. At the definitive procedure, 3% sodium tetradecyl sulfate was given into the trunk of the greater saphenous vein and into major varicosities. Some 0.75% and 0.3% sclerosant was also used but the total equated to 9.4 ml of 3% sodium tetradecyl sulfate. A class 2, full-length stocking was applied but severe pain 24 hours later caused removal of the stocking. Skin breakdown with abscess formation was noted in several places as seen in Figure 2. Hospitalization, intravenous antibiotics, and surgical debridement was required. The end result was unsatisfactory scar formation with hyperpigmentation (Fig. 5B).

If ultrasound-guided compression sclerotherapy is elected, appropriate patient selection must be done. Surgery and ultrasound-guided sclerotherapy are not interchangeable procedures and the latter should be reserved for carefully selected cases. The combination of poor patient selection, excessive doses of sclerosant in excess of the manufacturer's recommendations are commonly used in various places in the world and, in my opinion, this is a very dangerous combination indeed.

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